CORRESPONDENCE

- All letters must be typed with double spacing and signed by all authors.
- No letter should be more than 400 words.
- For letters on scientific subjects we normally reserve our correspondence columns for those relating to issues discussed recently (within six weeks) in the BMJ.
- We do not routinely acknowledge letters. Please send a stamped addressed envelope if you would like an acknowledgment.
- Because we receive many more letters than we can publish we may shorten those
 we do print, particularly when we receive several on the same subject.

Creutzfeldt-Jakob disease

SIR,—I am concerned at the apparent lack of up to date guidance from the Department of Health on sterilisation and disinfection procedures relating to patients with Creutzfeldt-Jakob disease. For some years autoclaving and disinfection with sodium hypochlorite have been the methods of choice. ^{1,3} Sodium hypochlorite is now thought to be only partially effective and has been superseded by isotonic sodium hydroxide. ^{1,5} A modified formalin mixture has also been recommended for the fixation of brains removed at necropsy. ⁵

Although information on these methods is available in at least one textbook, it does not seem to have been circulated in safety action bulletins or hazard notices.

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- 1 Gajdusek DC, Gibbs CJ, Asher DM, et al. Precautions in the medical care of, and in handling materials from, patients with transmissible virus dementia (Creutzfeldt-Jakob disease). N Engl J Med 1977;297:1253-8.
- 2 Brown P, Gibbs CJ, Amyx HL, et al. Chemical disinfection of Creutzfeldt-Jakob disease virus. N Engl J Med 1982;306: 1279-81.
- 3 Advisory Group on the Management of Patients with Spongiform Encephalopathy (Creutzfeldt-Jakob disease). Report. London: HMSO, 1981.
- 4 Brown P, Rohwer RG, Gajduseck DC. Sodium hydroxide decontamination of Creutzfeldt-Jakob disease virus. N Engl J Med 1984;320:727.
- 5 Committee on Health Care Issues of the American Neurological Association. Precautions in handling tissues, fluids and contaminating materials from patients with documented or suspected Creuztfeldt-Jakob disease. Ann Neurol 1986;19: 75-7.
- 6 Esiri MM, Oppenheimer DR. Diagnostic neuropathology. Oxford: Blackwell, 1989.

The toxic shock syndrome

SIR,—With reference to Dr Glyn R Williams's recent editorial on the toxic shock syndrome¹ we report on two teenage girls who died recently at this hospital after fulminating staphylococcal toxic shock syndrome related to using tampons. Both patients presented to their general practitioners with febrile illnesses of short duration in which diarrhoea was the dominant feature. Despite prompt referral to hospital and rapid diagnosis, aggressive medical treatment did not avert their deaths.

Discussion of these cases with our medical colleagues revealed a tendency to think of this condition as an American problem confined to the early 1980s. Clearly, this is not the case. Perhaps some doctors will be surprised to learn that these days many girls use tampons from the start of their menarche—one of our patients was only 14. Doctors treating any woman of menstrual age who

has febrile illness, especially one associated with diarrhoea and vomiting, should ask whether the patient is wearing a vaginal tampon. If so, it should be removed. This simple step might occasionally avert a more debilitating illness and very rarely save life.

Packets of tampons contain information sheets advising women on safe use. Yet our inquiries suggest that few women read these sheets and that most are unaware of potential problems. Parents, teachers, and doctors should advise young girls starting to use tampons how to do so safely.

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1 Williams GR. The toxic shock syndrome. Br Med J 1990;300: 960. (14 April.)

Calcium supplementation of the diet

SIR,—We were interested to learn that the calcium dinosaurs are not extinct and that Professors B E Christopher Nordin and Robert P Heaney were stimulated to reply¹ to our review of calcium nutrition.² The thrust of our review and the subsequent correspondence³ was to point out the pitfalls in interpreting studies of metabolic balance for calcium in judging the recommended daily allowance. From the evidence presented by Professors Nordin and Heaney it seems that the pitfalls are too well concealed.

Men adapt to changes in dietary intake of calcium.4 This adaptation may take several years to be complete. This phenomenon also occurs slowly in postmenopausal women.5 Thus the effect of a change in calcium nutrition induces transient changes until a new steady state comes about several years later. The steady state effect of altered calcium intake can be determined only from long term studies. There are still no adequately controlled studies to show whether an increase in calcium intake increases bone mass in the young independently of energy intake. Nor are there studies to show whether an increased calcium intake has any effect on skeletal consolidation or on subsequent risk of fracture after longitudinal growth has ceased. The oft cited study of Matkovic et al does not take account of differences in energy expenditure between the communities taking a high or low calcium diet.6

If we are to believe the conclusions of Professors Nordin and Heaney that "the [daily] allowance needed to meet this [calcium] requirement in normal subjects is about 800-1000 mg" we must conclude that the vast majority of the juvenile and young healthy adult population of the world is suffering from calcium deficiency. We must also

believe that most of the world population requires additional dietary calcium throughout life and we must consider the consequences this has for global nutritional policies. This view may depend largely on misinterpreting our existing knowledge. Perhaps we can stimulate Professors Nordin and Heaney to undertake the relevant studies which, if substantiating their hypothesis, would support more rationally their evangelical view.

Professors Nordin and Heaney may be surprised to learn that we are less at odds over the use of calcium in postmenopausal women, and had they read our review with care they would appreciate that we do not doubt that pharmacological doses of calcium delay the rate of bone loss at that time. A recent report emphasises the potential importance of this effect after surgical castration.7 Skeletal calcium losses are clearly attenuated by pharmacological manipulation of bone turnover. This suggests that calcium deficiency causes osteoporosis only in the same way that penicillin deficiency causes streptococcal infections. We would prefer, however, to consider both to be effective pharmacological interventions rather than causes of disease.

It might be expected that a decrease in the rate of bone loss would decrease the risk of osteoporotic fracture. This is probable but only inferred. The interpretation of the only studies directly examining the effect of calcium on fracture rates* is fatally flawed and we must await the outcome of adequately designed prospective studies. The search for the truth of tomorrow can perhaps be stimulated when we acknowledge that our conclusions of today have limitations.

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- 2 Kanis JA, Passmore R. Calcium supplementation of the diet. Br Med J 1989;298:137-40; 205-8.
- 3 Stevenson J. Calcium supplementation of the diet. Br Med J 1989;298:1034.
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- 6 Matkovic V, Kostial K, Simonovic I, Buzina R, Brodarec A, Nordin BEC. Bone status and fracture rates in two regions of Yugoslavia. Am J Clin Nutr 1979;32:540-9.
- 7 Stapan JJ, Pospichal J, Presl J, Pacovsky V. Prospective trial of ossein-hydroxyapatite compound in surgically induced postmenopausal women. *Bone* 1989;10:179-85.
- 8 Riggs BL, Seeman E, Hodgson SF, Taves DR, O'Fallon WM. Effect of the fluoride/calcium regimen on vertebral fracture occurring in postmenopausal osteoporosis. N Engl J Med 1982;306:446-50.
- Nordin BEC, Horsman A, Crilly AG, Marshall DH, Simpson M. Treatment of spinal osteoporosis in postmenopausal women. Br Med J 1980;280:451-4.